2026, Volume 3, Issue 6, Page N: 19-27. Impact Factor 5.381 ISSN (O): 3050-9491
ISSN (P): 3050-9483

World Journal of
Advance Multidisciplinary Research
www.wjamr.org

Research Article

CLINICO-ETIOLOGICAL PROFILE OF CHILDREN PRESENTING WITH GLOBAL DEVELOPMENTAL DELAY IN A TEACHING HOSPITAL OF RAJASTHAN: AN OBSERVATIONAL STUDY

Dr. Utkarsh Sharma[1], Dr. Priya Marwah[2]*, Dr. Rupesh Masand[3]

[1]Junior Resident, Department of Pediatrics, Mahatma Gandhi Medical College and Hospital.
[2]Professor & Unit Head, Department of Pediatrics, Mahatma Gandhi Medical College & Hospital.
ORCID ID: 0000-0001-7440-2502.
[3]Professor and Head, Department of Immunohematology and Blood Transfusion, Mahatma Gandhi Medical College & Hospital.

Article Info

Article Received: 05 June 2026,

Article Revised: 25 June 2026,

Published on: 01 July 2026

***Corresponding author:**

Dr. Priya Marwah

Professor & Unit Head, Department of Pediatrics, Mahatma Gandhi Medical College & Hospital.
ORCID ID: 0000-0001-7440-2502.
priyamarwah21@gmail.com

https://doi.org/10.5281/zenodo.21100047

How to cite this Article:
Dr. Utkarsh Sharma[1], Dr. Priya Marwah[2]*, Dr. Rupesh Masand[3] (2026). Clinico-Etiological Profile of Children Presenting With Global Developmental Delay In A Teaching Hospital of Rajasthan: An Observational Study. World Journal of Advance Multidisciplinary Research, 3(6), 19–27.

ABSTRACT

Background: Global Developmental Delay (GDD) is one of the most common neurodevelopmental conditions encountered in paediatric practice, defined as significant delay in two or more developmental domains in children typically under the age of five years. The aim of this study was to determine the clinical and etiological profile of children presenting with GDD and to study associated co-morbidities such as convulsions, cerebral palsy and behaviour disorders. **Methodology:** A hospital-based observational study was conducted at the Department of Paediatrics, Mahatma Gandhi Medical College and Hospital, Jaipur, Rajasthan, from April 2024 to September 2025. A total of 100 children aged 6 months to 5 years presenting with GDD were enrolled after obtaining informed parental consent. Developmental assessment was performed using the Trivandrum Developmental Screening Chart (TDSC) and children with a developmental quotient (DQ) less than 70% in two or more domains were included. Statistical analysis was performed using SPSS version 20.0; quantitative data were expressed as mean ± SD and qualitative data as percentages and proportions. **Results:** The majority of children belonged to the 3–4 years age group (50%) with a mean age of 3.647 ± 1.541 years. Male predominance was observed (77% males vs. 23% females). Consanguinity was present in only 5% and positive family history in 7% of cases. The most common presenting symptom was isolated developmental delay (40%), followed by developmental delay with seizures (15%) and feeding problems (15%). On clinical examination, cerebral palsy was the most common CNS finding (20%), followed by microcephaly (15%). Most children had a DQ in the range of 21–30% (44%), reflecting moderate to severe developmental impairment. Perinatal asphyxia was the leading etiology (40%), followed by idiopathic causes (21%) and cortical dysgenesis (10%). Neuroimaging was abnormal in 28% and electrophysiological studies were abnormal in 54% (EEG 34%, BERA 20%). Anemia was the most common laboratory finding (30%). Neurological co-morbidities included sleep issues (30%), seizures (25%), cerebral palsy (20%) and feeding problems (20%). Non-neurological co-morbidities included anemia (30%), undernutrition (25%) and constipation (25%). Psychiatric/behavioral co-morbidities included mood disorders (15%), ADHD (10%) and autism (7%). **Conclusion:** Perinatal asphyxia is the predominant and largely preventable cause of GDD in this region. Most children presented late with moderate to severe developmental delay, underscoring the need for early developmental screening and timely referral. The high burden of nutritional co-morbidities, particularly anemia and undernutrition, highlights the importance of integrated maternal and child health programmes. Improved antenatal and perinatal care, neonatal resuscitation services and early multidisciplinary intervention are essential to reduce the burden of GDD.

KEYWORDS: Global Developmental Delay, Perinatal Asphyxia, Developmental Quotient, Clinico-etiological Profile, Cerebral Palsy, Rajasthan.

INTRODUCTION

Global developmental delay (GDD) is a multifaceted neurodevelopmental condition observed in young children, typically those under the age of five years, where there is significant lag in achieving expected milestones across multiple domains of development.[1] This includes delays in gross motor skills such as rolling over, sitting unsupported, crawling and walking; fine motor abilities like grasping objects, transferring items between hands and manipulating small toys; speech and language development both expressive and receptive; cognitive functions including problem-solving, memory and learning; social-personal interactions such as eye contact, joint attention and responding to name; and adaptive behaviors such as daily self-care activities.[1,2] The diagnosis is established when performance falls at least two standard deviations below age-matched norms on validated developmental screening tools, such as the Bayley Scales of Infant and Toddler Development or the Denver Developmental Screening Test.[3,4] GDD is one of the most common conditions encountered in paediatric practice. The estimated global prevalence ranges from 1% to 3% of children under 5 years of age, making it a significant public health concern.[5,6] Pooled prevalence data from low- and middle-income countries (LMICs) is considerably higher; a 2024 meta-analysis reported a pooled prevalence of 18.83% of confirmed developmental delay among children in LMICs, with the highest rates in Africa reaching 26.69%.[7] GDD disproportionately affects male children across most published cohorts.[8]

The etiology of GDD is heterogeneous, encompassing prenatal causes such as genetic/chromosomal disorders, congenital infections, brain malformations and metabolic disorders; perinatal causes including hypoxic-ischemic encephalopathy (HIE), prematurity, neonatal seizures and bilirubin encephalopathy; postnatal causes such as CNS infections, trauma and nutritional deprivation; and a substantial proportion of idiopathic cases where no definite cause is identified.[2,3] Genetic factors predominate (up to 50%), followed by perinatal insults with 30–50% initially idiopathic pending advanced testing like exome sequencing.[3,9,10,11] In resource-constrained settings like India, perinatal insults and nutritional deficiencies are disproportionately prevalent and constitute major modifiable risk factors. In India, several studies have highlighted perinatal asphyxia as the leading preventable cause of GDD, with significant contributions from genetic disorders, cortical dysgenesis, congenital infections and metabolic disorders.[12,13] The clinico-etiological profile varies substantially across regions depending on the availability of diagnostic facilities, sociocultural factors, degree of consanguinity and standards of antenatal and perinatal care.[14,15] Despite this, data from Rajasthan specifically describing the clinical and etiological spectrum of GDD in children remain limited.

The clinico-etiological profile of children with GDD holds paramount significance in modern paediatric neurology and developmental medicine, as it facilitates timely etiological diagnosis, initiation of specific therapies and multidisciplinary interventions that optimize neuroplasticity during critical early developmental windows.[16,17] Delineating local profiles empowers evidence-based guidelines, reduces diagnostic odysseys for families, improves long-term quality of life and alleviates socioeconomic burdens associated with lifelong support needs.

METHODOLOGY

This was a hospital-based observational study conducted at the Department of Paediatrics, Mahatma Gandhi Medical College and Hospital (MGMCH), Jaipur, Rajasthan from April 2024 to September 2025. Based on published data (Koul et al., 2012)[18] reporting 71.8% etiological yield in GDD and at 95% confidence level with 10% relative allowable error, a minimum of 95 children were required; the sample size was rounded to 100. Children aged 6 months to 5 years with GDD were included. Children outside this age range or whose parents refused consent were excluded.

Developmental Assessment

Developmental assessment was performed using the Trivandrum Developmental Screening Chart (TDSC), a validated Indian tool covering gross motor, fine motor, language and social domains. Children with a developmental quotient (DQ) less than 70% in two or more domains were confirmed to have GDD. Corrected gestational age was applied for premature infants.

Clinical Evaluation and Investigations

All enrolled children underwent detailed clinical history (antenatal, perinatal and postnatal), general and neurological examination including anthropometry, CNS findings and systemic assessment. Baseline investigations included hemoglobin estimation, thyroid function tests and CMV serology. Further investigations such as neuroimaging (MRI/CT brain), EEG, BERA, karyotyping, metabolic screening and biotin response were performed based on clinical indications. Etiological classification was done into perinatal, genetic, metabolic, infectious, structural and idiopathic categories. Associated co-morbidities were assessed across neurological, non-neurological and psychiatric/behavioral domains.

Statistical Analysis

Quantitative data were expressed as mean ± SD and qualitative data as percentages and proportions. Chi-square tests and Z-scores were used for comparison of categorical variables; $p < 0.05$ was considered statistically significant. All analyses were performed using SPSS version 20.0.

RESULTS

A total of 100 children with Global Developmental Delay (GDD) were enrolled in this study. The findings are presented below under demographic characteristics and patient profile, clinical examination findings, investigation profile, etiological classification and associated co-morbidities.

Table 1: Demographic and Patient Characteristics of Children with GDD (n=100)

Parameter	Frequency (n)	Percentage (%)
Age Group		
<1 year	5	5%
1–2 years	10	10%
2–3 years	20	20%
3–4 years	50	50%
4–5 years	15	15%
Mean ± SD	3.647 ± 1.541 years	
Gender		
Male	77	77%
Female	23	23%
Consanguinity		
Present	5	5%
Absent	95	95%
Family History		
Positive	7	7%
Negative	93	93%
Presenting Symptoms		
Isolated Developmental Delay (DD)	40	40%
DD with Seizures	15	15%
DD with Sleep Problems	10	10%
DD with Abnormal Behaviour	5	5%
DD with Feeding/Eating Problem	15	15%
DD with Abnormal Movement	5	5%
DD with Poor Weight Gain	10	10%

The majority of children belonged to the 3–4 years age group (50%) with a mean age of 3.647 ± 1.541 years. A clear male predominance was observed with 77% males compared to 23% females. Consanguinity was present in only 5% of cases and positive family history in only 7%, indicating that most cases were sporadic in nature. The most common presenting symptom was isolated developmental delay (40%), followed by developmental delay with seizures (15%) and feeding/eating problems (15%). Sleep problems and poor weight gain were observed in 10% each, while abnormal behaviour and abnormal movements were the least common, each seen in 5% of children.

Table 2: Antenatal, Perinatal and Postnatal Risk Factor Profile (n=100).

Risk Factors	Frequency (n)	Percentage (%)
Antenatal		
Diabetes mellitus	5	5%
Hypertension	8	8%
Infection	10	10%
None	77	77%
Perinatal		
Birth asphyxia	40	40%
Respiratory distress	10	10%
Seizures	15	15%
Bilirubin encephalopathy	3	3%
Septicemia	5	5%
Prematurity / VLBW	10	10%
None	17	17%
Postnatal		
Sequelae of CNS infections	7	7%

Risk Factors	Frequency (n)	Percentage (%)
None	93	93%

The majority of mothers (77%) had no significant antenatal complications. Among identifiable antenatal risk factors, infections were the most common (10%), followed by hypertension (8%) and diabetes (5%). Perinatal risk factors were highly prevalent; birth asphyxia was the most common, seen in 40% of children, followed by neonatal seizures (15%), respiratory distress (10%) and prematurity/very low birth weight (10%). Only 17% of children had no perinatal complications, highlighting the major contribution of birth-related insults. Postnatal factors were minimal with sequelae of CNS infections in only 7% of cases, indicating that postnatal causes were less significant than perinatal factors in this cohort.

Table 3: Clinical Examination Findings and Developmental Quotient Distribution (n=100).

Examination Findings	Frequency (n)	Percentage (%)
General Examination		
Multiple congenital anomalies	5	5%
Cutaneous markers for NCS	3	3%
Skin/Hair abnormalities	10	10%
None	82	82%
CNS Examination		
Microcephaly	15	15%
Abnormal movements	6	6%
Cerebral palsy	20	20%
None	59	59%
Other Systems		
Congenital heart disease	5	5%
Hepatosplenomegaly	3	3%
None	92	92%
Developmental Quotient (DQ)		
≤20%	14	14%
21–30%	44	44%
31–40%	9	9%
41–50%	19	19%
51–60%	8	8%
61–70%	6	6%

Most children (82%) had no significant abnormalities on general examination. Among those with findings, skin and hair abnormalities were seen in 10%, multiple congenital anomalies in 5% and cutaneous markers of neurocutaneous syndromes in 3%, suggesting underlying syndromic or genetic conditions in a subset. On CNS examination, 41% of children had identifiable findings; cerebral palsy was the most common (20%), followed by microcephaly (15%) and abnormal movements (6%). Systemic involvement was uncommon with congenital heart disease in 5% and hepatosplenomegaly in 3%. The majority of children had severely impaired developmental quotients; 44% had DQ in the 21–30% range, followed by 19% in the 41–50% range and 14% with DQ ≤20%. Overall, 81% of children had DQ ≤40%, reflecting predominantly moderate to severe developmental impairment at the time of presentation.

Table 4: Investigation Profile of Children with GDD (n=100).

Investigation	Frequency (n)	Percentage (%)
Blood Investigations		
Anemia	30	30%
IgM against CMV / Urine CMV positive	5	5%
Hypothyroidism	3	3%
Normal	62	62%
Neuroimaging (MRI/CT)		
Abnormal	28	28%
Normal	72	72%
Electrophysiology		
EEG abnormal	34	34%

BERA abnormal	20	20%
None abnormal	46	46%
Other Investigations		
Karyotyping abnormality	4	4%
Dramatic response to Biotin therapy	2	2%
None	94	94%

Blood investigations were normal in 62% of children; anemia was the most common abnormality, seen in 30% of cases, making it the most frequent and potentially treatable associated finding. Congenital cytomegalovirus infection was detected by IgM serology and urine positivity in 5% and hypothyroidism in 3%. Neuroimaging was abnormal in 28% and normal in 72%, suggesting a moderate diagnostic yield of structural imaging in this cohort. Overall, 54% of children had electrophysiological abnormalities; EEG was abnormal in 34% and BERA in 20%, indicating a high prevalence of subclinical neurological and auditory dysfunction. Karyotyping revealed chromosomal abnormalities in 4% of children and dramatic response to biotin therapy, suggestive of biotin deficiency, was observed in 2%, representing important treatable and genetically identifiable causes.

Table 5: Etiological Profile of Children with GDD (n=100).

Etiology	Frequency (n)	Percentage (%)
Perinatal asphyxia	40	40%
Idiopathic	21	21%
Cortical dysgenesis	10	10%
Sequelae of acute meningoencephalitis	7	7%
Congenital cytomegalovirus (CMV) infection	5	5%
Genetic (Trisomy)	4	4%
Congenital hypothyroidism	3	3%
Bilirubin encephalopathy	3	3%
Neurocutaneous syndromes	3	3%
Infantile Tremor Syndrome (ITS)	2	2%
Biotin deficiency	2	2%
Total	100	100%

Perinatal asphyxia was the most common etiology, accounting for 40% of all cases, highlighting its major and preventable contribution to the burden of GDD. Idiopathic cases constituted 21%, where no definite cause could be identified despite detailed evaluation. Cortical dysgenesis accounted for 10% of cases, while sequelae of acute meningoencephalitis contributed 7%. Congenital CMV infection was identified in 5%, genetic causes (trisomy) in 4%, congenital hypothyroidism in 3%, bilirubin encephalopathy in 3%, neurocutaneous syndromes in 3% and both infantile tremor syndrome and biotin deficiency in 2% each. The distribution demonstrates that while perinatal causes dominate, a diverse range of prenatal, genetic, metabolic, infectious and structural factors also contribute to the etiological spectrum of GDD in this population.

Table 6: Associated Co-morbidities in Children with GDD (n=100).

Co-morbidity	Frequency (n)	Percentage (%)
Neurological		
Sleep issues	30	30%
Seizures	25	25%
Cerebral palsy	20	20%
Feeding problems	20	20%
Impaired vision	12	12%
Impaired hearing	8	8%
Non-neurological		
Anemia	30	30%
Undernutrition	25	25%
Constipation	25	25%
Respiratory tract infections (RTI)	20	20%
Psychiatric / Behavioral		
Mood disorders	15	15%
ADHD	10	10%

Autism spectrum disorder	7	7%
Aggressive behaviour	5	5%
None	63	63%

Neurological co-morbidities were highly prevalent. Sleep issues were the most common, affecting 30% of children, followed by seizures in 25%, cerebral palsy in 20%, feeding problems in 20%, impaired vision in 12% and impaired hearing in 8%. Among non-neurological co-morbidities, anemia was the most common, observed in 30% of children, followed by undernutrition and constipation in 25% each and respiratory tract infections in 20%. These findings highlight the significant systemic and nutritional burden alongside neurological dysfunction in children with GDD. Regarding psychiatric and behavioral co-morbidities, the majority of children (63%) had no psychiatric or behavioral issues. Among those affected, mood disorders were the most frequent (15%), followed by ADHD (10%), autism spectrum disorder (7%) and aggressive behaviour (5%), indicating that a notable subset of GDD children have concurrent psychiatric conditions requiring early recognition and intervention.

DISCUSSION

Global Developmental Delay (GDD) represents a heterogeneous group of disorders characterized by significant delay in two or more developmental domains including motor, language, cognitive and social functions. The present study provides a comprehensive analysis of 100 children with GDD presenting to a tertiary care teaching hospital in Rajasthan and compares findings with published national and international literature. In the present study, the majority of children (50%) belonged to the 3–4 years age group with a mean age of 3.647 ± 1.541 years. This relatively late age at presentation suggests a delay in recognition or referral for developmental concerns, which is a commonly observed pattern in developing countries with limited primary-level developmental surveillance. Singh et al. (2023)[19] from Eastern India reported a mean age of 4.1 years, Alamri et al. (2021)[20] reported a mean of 37.9 months and Phayde et al. (2025)[21] reported a median age of 3.3 years, all consistent with the present finding. In contrast, Sharma et al. (2023)[22] from Maharashtra reported a median age of only 15.5 months and Tikaria et al. (2010)[23] from AIIMS New Delhi reported 23.6 months, indicating earlier identification in more specialized referral settings. Xiong et al. (2025)[24] confirmed in a large-scale study of 32,511 children that multi-domain severity of GDD becomes more apparent with increasing age. The older age at presentation in this cohort likely reflects limited awareness of early developmental surveillance and restricted access to specialized paediatric services in Rajasthan.

A clear male predominance was observed in the present study with 77% males compared to 23% females. This is consistent with the well-established pattern reported across multiple studies; Sharma et al. (2023)[22] reported 69.2% males, Singh et al. (2023)[19] reported 72.9% and Gupta et al. (2025)[25] found 62.22% male predominance. The higher male preponderance may be explained by the biological vulnerability of males to neurodevelopmental disorders and X-linked genetic conditions, as well as socio-cultural factors leading to preferential healthcare-seeking behaviour for male children in this region. Consanguinity was present in only 5% of cases, notably lower than Habibullah et al. (2019[26]; 57%), Sharma et al. (2023[22]; 42.3%) and Nelanuthala et al. (2021[27]; 54%), indicating that consanguinity-related genetic risk was not a major contributory factor in this cohort. Similarly, positive family history was present in only 7% of cases, suggesting predominantly sporadic rather than familial etiology.

The majority of mothers (77%) had no significant antenatal history. Among identifiable antenatal risk factors, maternal infections were the most common (10%), followed by hypertension (8%) and diabetes (5%), consistent with Habibullah et al. (2019)[26] who reported 84% of children having no antenatal complications. Perinatal risk factors were highly significant; birth asphyxia was the most common, seen in 40% of children, followed by neonatal seizures (15%), respiratory distress (10%) and prematurity/VLBW (10%). Only 17% had no perinatal complications, reinforcing the predominant contribution of birth-related insults. These findings are consistent with Gupta et al. (2025[25]; 46.67%), Phayde et al. (2025[21]; 33.6%) and Bandara et al. (2020[28]; 27.6%). Aldosari et al. (2024)[2] confirmed that premature birth and low birth weight increase neurodevelopmental impairment risk by 3–5 fold. Postnatal causes were minimal with CNS infection sequelae in 7%, comparable to Bandara et al. (2020[28]; 7%) and Ozmen et al. (2005[29]; ~7%).

Most children (82%) had no significant findings on general examination. Among those with findings, skin and hair abnormalities were seen in 10%, multiple congenital anomalies in 5% and cutaneous markers of neurocutaneous syndromes in 3%, suggesting underlying syndromic or genetic conditions in a subset. On CNS examination, cerebral palsy was the most common finding (20%), followed by microcephaly (15%) and abnormal movements (6%), consistent with Aggarwal et al. (2013)[30] and Bandara et al. (2020).[28] Systemic involvement was uncommon with congenital heart disease in 5% and hepatosplenomegaly in 3%. Regarding developmental quotient, the majority of children had severely impaired DQ; 44% had DQ in the 21–30% range and 81% had DQ ≤40%, reflecting predominantly moderate to severe developmental impairment at presentation. This is closely comparable to the mean DQ of 29.75 ± 17.8 reported by Aggarwal et al. (2013)[30] and

underscores the need for early identification before children reach the preschool age.

Blood investigations were normal in 62% of children. Anemia was the most common laboratory abnormality (30%), making nutritional deficiency the most frequent and potentially modifiable co-existing condition. Congenital CMV was detected in 5% and hypothyroidism in 3%. Neuroimaging was abnormal in 28%, which is lower than rates reported by Gupta et al. (70%), Alamri et al.[20] (70%), Ali et al.[31] (68%) and Phayde et al.[21] (75.7%). This difference may reflect the fact that neuroimaging was performed based on clinical indications rather than universally, unlike microcephaly- or seizure-focused cohorts. Electrophysiological studies revealed abnormalities in 54% (EEG 34%, BERA 20%), highlighting a high prevalence of subclinical neurological and auditory dysfunction. This is consistent with Habibullah et al. (2019[26]; 45% EEG), Lopes et al. (2025[32]; 56% EEG) and Aggarwal et al. (2013[30]; 16.9% hearing abnormalities). Karyotyping revealed chromosomal abnormalities in 4% and dramatic response to biotin therapy was observed in 2%, representing important treatable causes identified in this cohort.

Perinatal asphyxia was the leading etiology (40%), followed by idiopathic causes (21%), cortical dysgenesis (10%) and sequelae of meningoencephalitis (7%). Sachdeva et al. (2019)[33] from SMS Medical College, Jaipur, reported perinatal insult as the most common cause in 63.1% of GDD children, closely consistent with the present study. Gupta et al. (2025)[25] reported 46.67% and Bandara et al. (2020)[28] reported HIE in 27.6%. The high idiopathic proportion (21%) is consistent with global estimates of 30-50% and likely reflects limited availability of chromosomal microarray and whole-exome sequencing in this setting. Sharma et al. (2023)[22] from Maharashtra with access to advanced genetic testing, identified prenatal/genetic causes in 50% with an overall etiological yield of 88.4%, illustrating the potential for significantly improved diagnostic accuracy with expanded resources. Congenital CMV (5%), trisomy (4%), congenital hypothyroidism (3%), bilirubin encephalopathy (3%) and infantile tremor syndrome and biotin deficiency (2% each) reflect important albeit less common etiologies in this cohort.

Neurological co-morbidities were highly prevalent; sleep issues (30%), seizures (25%), cerebral palsy (20%), feeding problems (20%), impaired vision (12%) and hearing impairment (8%). Seizure prevalence was somewhat lower than Gupta et al. (2025[25]; 51.11%) and Albaradie et al. (2021[34]; 56%), possibly reflecting differences in patient selection. Non-neurological co-morbidities were also markedly prevalent; anemia (30%), undernutrition and constipation (25% each) and RTI (20%). Gupta et al. (2025)[25] reported pallor in 93.33% and malnutrition in 82.22%, higher than the present study, while Phayde et al. (2025)[21] reported 58.7% malnutrition. These findings collectively highlight the significant systemic and nutritional burden co-existing alongside neurological dysfunction in children with GDD. Psychiatric and behavioral co-morbidities were present in 37% of children; mood disorders (15%), ADHD (10%), ASD (7%) and aggressive behaviour (5%). This is lower than the 62.3% ASD prevalence reported by Shan et al. (2022)[35] and the 26.2% ASD etiology rate by Singh et al. (2023)[19], which may reflect differences in patient selection, use of formal autism diagnostic tools and the cross-sectional nature of the present study.

CONCLUSION

The present study shows that Global Developmental Delay (GDD) commonly presents in early childhood, especially between 3-4 years indicating delayed diagnosis. There was a clear male predominance, while consanguinity and family history were low, suggesting limited genetic contribution. Most children presented with isolated developmental delay, though neurological symptoms such as seizures and feeding problems were also observed. Perinatal factors, particularly birth asphyxia, were the leading cause, followed by idiopathic and structural causes, highlighting the importance of preventable factors. Neurological abnormalities like cerebral palsy and microcephaly were common and most children had moderate to severe developmental delay, reflecting late identification. Investigations revealed moderate neuroimaging yield and significant electrophysiological abnormalities, while anemia was the most common laboratory finding. Co-morbidities were mainly neurological (seizures, sleep issues) and nutritional (anemia, undernutrition) with relatively fewer psychiatric or behavioral problems. Overall, the study emphasizes that GDD is largely associated with preventable perinatal insults and nutritional deficiencies, highlighting the need for early screening, improved maternal and neonatal care and timely multidisciplinary intervention to improve outcomes.

REFERENCES

1. Khan I, Leventhal BL. Developmental delay (2023). Available at: https://www.ncbi.nlm.nih.gov/books/NBK562231/
2. Aldosari AN, Aldosari TS. Comprehensive evaluation of the child with global developmental delays or intellectual disability. Clinical and Experimental Pediatrics. 2024 May 29; 67(9): 435.
3. Sharma AR, Siddiqui MS, Magar S, Kale A, Nelanuthala M, Singh SP, Sharma A. The etiological profile of global developmental delay at a tertiary care hospital in India: An observational study. Cureus. 2023 Jun 28; 15(6).
4. Mithyantha R, Kneen R, McCann E, Gladstone M. Current evidence-based recommendations on investigating children with global developmental delay. Archives of disease in childhood. 2017 Nov 1; 102(11): 1071-6.

5. Michelson DJ, Shevell MI, Sherr EH, Moeschler JB, Gropman AL, Ashwal S. Evidence Report: Genetic and metabolic testing on children with global developmental delay [RETIRED] Report of the Quality Standards Subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society. Neurology. 2011 Oct 25; 77(17): 1629-35.
6. Juneja M, Gupta A, Sairam S, Jain R, Sharma M, Thadani A, Srinivasan R, Lingappa L, Ahmed S, Multani KS, Buch P. Diagnosis and management of global development delay: consensus guidelines of growth, development and behavioral pediatrics chapter, neurology chapter and neurodevelopment pediatrics chapter of the Indian Academy of Pediatrics. Indian pediatrics. 2022 May; 59(5): 401-15.
7. Moeschler JB, Shevell M, Genetics CO, Saul RA, Chen E, Freedenberg DL, Hamid R, Jones MC, Stoler JM, Tarini BA. Comprehensive evaluation of the child with intellectual disability or global developmental delays. Pediatrics. 2014 Sep; 134(3): e903.
8. Flore LA, Milunsky JM. Updates in the genetic evaluation of the child with global developmental delay or intellectual disability. Inseminars in pediatric neurology 2012 Dec 1; 19(4): 173-180. WB Saunders.
9. Wikipedia. Global Developmental Delay. Available at: https://en.wikipedia.org/wiki/Global_developmental_delay.
10. Jimenez-Gomez A, Standridge SM. A refined approach to evaluating global developmental delay for the international medical community. Pediatric neurology. 2014 Aug 1; 51(2): 198-206.
11. Geeky Medics. Available at: https://geekymedics.com/global-developmental-delay/
12. Sachdeva P, Sitaraman S, Minocha P, Jain R, Choudhary A, Jetha K. Etiological profile of children with global developmental delay: a single center study. Journal of Pediatric Neurology. 2019 Jun; 17(03): 111-7.
13. Sharma AR, Siddiqui MS, Magar S, Kale A, Nelanuthala M, Singh SP, Sharma A. The etiological profile of global developmental delay at a tertiary care hospital in India: An observational study. Cureus. 2023 Jun 28; 15(6).
14. Jimenez-Gomez A, Standridge SM. A refined approach to evaluating global developmental delay for the international medical community. Pediatric neurology. 2014 Aug 1; 51(2): 198-206.
15. Srour M, AlHakeem A, Shevell M. Global developmental delay and intellectual disability. InRosenberg's molecular and genetic basis of neurological and psychiatric disease 2020 Jan 1; 269-281. Academic Press.
16. Tirosh E, Jaffe M. Global developmental delay and mental retardation—a pediatric perspective. Developmental disabilities research reviews. 2011 Nov; 17(2): 85-92.
17. Rodan LH, Stoler J, Chen E, Geleske T. Genetic evaluation of the child with intellectual disability or global developmental delay: clinical report. Pediatrics. 2025 Jul 1; 156(1): e2025072219.
18. Koul R, Al-Yahmedy M, Al-Futaisi A. Evaluation children with global developmental delay: A prospective study at Sultan Qaboos University Hospital, Oman. Oman medical journal. 2012 Jul; 27(4): 310.
19. Singh A, Roy S, Kumar S, Ghosh S. Clinical and Etiological Profile of Children with Developmental Delay: An Observational Cross - Sectional Study from Eastern India. IJSR. 2023 April; 12(4): 877-881.
20. Alamri A, Aljadhai YI, Alrashed A, Alfheed B, Abdelmoaty R, Alenazi S, Alhashim A, Benini R. Identifying clinical clues in children with global developmental delay/intellectual disability with abnormal brain magnetic resonance imaging (MRI). Journal of Child Neurology. 2021 May; 36(6): 432-9.
21. Phayde SS, Mundkur SC, Pereira RA, Lewis LE, Hebbar SA, Bhat R, Kini P, Moras KJ. Clinicoradiological profile of children presenting with global developmental delay and microcephaly. Clinical Epidemiology and Global Health. 2025 Jul 1; 34: 102076.
22. Sharma AR, Siddiqui MS, Magar S, Kale A, Nelanuthala M, Singh SP, Sharma A. The etiological profile of global developmental delay at a tertiary care hospital in India: An observational study. Cureus. 2023 Jun 28; 15(6).
23. Tikaria A, Kabra M, Gupta N, Sapra S, Balakrishnan P, Gulati S, Pandey RM, Gupta AK. Aetiology of global developmental delay in young children: Experience from a tertiary care centre in India. National Medical Journal of India. 2010 Jan 1; 23(6): 324.
24. Xiong NN, Cui ZJ, Zhao M, Du J, Li SJ, Li MH, Lu YY, Liang AM, Ma Y. Analysis of developmental function in 32 511 children with global developmental delay. Zhonghua liu xing bing xue za zhi= Zhonghua liuxingbingxue zazhi. 2025 Jun 10; 46(6): 1051-7.
25. Gupta M, Chandra S, Negi V. Unravelling Global Developmental Delay: Risk Factors and Comorbidities in Young Children at a Tertiary Care Hospital. International Journal of Medical Research. 2025 Jul 1; 13(03): 35-45.
26. Habibullah H, Albradie R, Bashir S. Identifying pattern in global developmental delay children: A retrospective study at King Fahad specialist hospital, Dammam (Saudi Arabia). Pediatric Reports. 2019 Dec 2; 11(4): 8251.
27. Nelanuthala M, Kaur B, Ingale V, Magar S, Joshi P. Having a common ancestor; significance of consanguinity and genetic diseases. Int J Contemp Pediatr. 2021; 8(1): 26-9.
28. Bandara U. Aetiological profile of global developmental delay in children under 5 years of age: experience from a tertiary referral centre. Sri Lanka Journal of Child Health. 2020 Dec 5.
29. Ozmen M, Tatli B, Aydinli N, Çalişkan M, Demirkol M, Kayserili H. Etiologic evaluation in 247 children with global developmental delay at Istanbul, Turkey.

Journal of tropical pediatrics. 2005 Oct 1; 51(5): 310-3.
30. Aggarwal A, Mittal H, Patil R, Debnath S, Rai A. Clinical profile of children with developmental delay and microcephaly. Journal of neurosciences in rural practice. 2013 Jul; 4(3): 288.
31. Ali AS, Syed NP, Murthy GS, Nori M, Abkari A, Pooja BK, Venkateswarlu J. Magnetic resonance imaging (MRI) evaluation of developmental delay in pediatric patients. Journal of clinical and diagnostic research: JCDR. 2015 Jan 1; 9(1): TC21.
32. Lopes MG, Alves AC, Pedrosa I, Lopes C, Lemos A, Pereira E, Henriques M. Screening, Diagnosis and Investigation of Global Developmental Delay and Intellectual Developmental Disorder. Cureus., 2025 Dec 12; 17(12).
33. Sachdeva P, Sitaraman S, Minocha P, Jain R, Choudhary A, Jetha K. Etiological profile of children with global developmental delay: a single center study. Journal of Pediatric Neurology. 2019 Jun; 17(03): 111-7.
34. Albaradie R, Habibullah H, Mir A, Alshammari AK, Alajmi MS, Alsubaie FA, Alsudairi RR, Bashir S. The prevalence of seizures in children with developmental delay. Neurosciences Journal. 2021 Apr 1; 26(2): 186-91.
35. Shan L, Feng JY, Wang TT, Xu ZD, Jia FY. Prevalence and developmental profiles of autism spectrum disorders in children with global developmental delay. Frontiers in Psychiatry. 2022 Jan 18; 12: 794238.